9) Cardiac Ventricular Hypertrophy in M-SHRSP. Jun Yamamoto, Masateru Minoda and Tsuneyuki Suzuki. Department of Pathology, Kinki University School of Medicine, Osaka-fu 589.

INTRODUCTION: M-SHRSP (Okamoto et al, 1984), in addition to severe hypertension and cerebral stroke, also exhibit cardiac hypertrophy. In this study we morphologically and microphotometrically examined the relationships between blood pressure and cardiac hypertrophy.

MATERIALS AND METHODS: Male M-SHRSP (F_3 - F_{20}) aged 30, 60, 90, 120 and 150 days, and aged-matched WKY as controls were used. Blood pressures (BP) were measured using the tail-pulse pickup method without anesthesia. Hearts were removed, weighed and fixed in 10% formalin soon after natural death or sacrifice. Examination included: 1) Muscle fiber bundles were examined using a pincette to strip the fibers starting from the surface of the ventricles. 2) Following HE and PTAH staining, coronal and longitudinal sections were taken from the middle and the apex of the ventricles. For each layer examined in 1), above, the transverse diameters of 100 muscle cells and the lengths (distances between two intercalated discs) of 50 muscle cells were measured using an image analysis system (Kontron MOP-20). 3) Left ventricle (LV) cross section wall thicknesses and areas were measured. 4) Again, for each layer examined in 1), above, the nuclear DNA content of 50 muscle cells stained with pararosaniline-Feulgen reaction were measured using a fluorescence microscope (Nikon VFS-R) and a cytofluorometer (Nikon PI). 5) The numbers of myocardial fascicle and fiber disarrays were measured using HE-stained mid-ventricle coronary sections.

RESULTS AND DISCUSSIONS: BP increased rapidly in M-SHRSP, being about 160 mmHg at 6 weeks and 250 mmHg at 12 weeks. M-SHRSP heart weights were greater than those of WKY after 60 days of age. At 150 days there were 1.3 times those of WKY. LV walls and septums (S) were divided into three layers and the right ventricle (RV) walls were divided into two. In the walls surrounding the LV, the middle layer started from the annulus fibrosus and descended counterclockwise spirally, then joined into the deep layer at the apex. The deep layer ran counterclockwise annularly to the annulus fibrosus. For M-SHRSP and WKY at 30 days of age, there were no significant differences in cardiac muscle diameters (MCD), in cardiac muscle lengths (MCL) nor in cardiac muscle cell nuclear DNA content. At 60 days of age, these figures, with the exception of the RV in M-SHRSP, increased in all walls. These increases accorded with rapid BP elevations. Also, at this age, MCD increases in the middle and deep layers of the lateral wall of the LV were most remarkable. It is thought that these layers are located in the center of the free wall of the LV and are more easily effected by hypertensive LV pressure elevations than are the other layers. After 90 days, MCDs and MCLs in the middle and deep layers of the LV walls increased equally. This means that these layers are proportionately acted upon by LV pressure. At 60 days, cardiac muscle cell nuclear DNA content in the middle and deep layers of the walls around the LV also increased equally. This preceded the increases in MCDs and MCLs found in M-SHRSP. At 90 days of age, RV superficial and deep layer MCDs, MCLs and cardiac muscle cell nuclear DNA content also increased. However, from then on these tended to be similar to the findings from the LV superficial layer. This appears to be a secondary effect of LV hypertrophy. At each age level in M-SHRSP the thickness of the S was less than that of the free LV wall. Between M-SHRSP and WKY, at none of the age levels, were any significant differences in LV cross-section area found. This means that the M-SHRSP hearts showed typical concentric hypertrophy. There were many myocardial fascicle and fiber disarrays in the LV middle and deep layers in both M-SHRSP and WKY. With aging, these increased more rapidly in M-SHRSP than they did in WKY. From this it can be concluded that LV pumping action is handled primarily by the LV wall middle and deep layers. In these layers in M-SHRSP cardiac muscle cell and nuclei hypertrophy were remarkable and, accordingly, there was an increase in myocardial fascicle and fiber disarray.